THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES.

OCTOBER, 1891.

ON THE DISEASES OF THE KIDNEYS POPULARLY CALLED "BRIGHT'S DISEASE."

By Francis Delafield, M.D., LL.D.,
of New York.

Our notions concerning the nature of diseases and their treatment are greatly influenced by names and classifications. So, as our knowledge becomes more accurate, it is important, as fast as may be, to modify and change such names and classifications as have become a hindrance instead of a help.

The ordinary history of such names is that during the earlier periods of medicine some man more able than his fellows was able to group together a number of cases of diseases, to describe them plainly and to venture some theory as to their nature. Such a group of diseases then became known by the name of its describer, or by some arbitrary term. As time went on such a name acquired the dignity of use and custom, became generally accepted, and more and more traditions and classical descriptions accumulated about it, until in men's minds were established a name and a clinical picture to which, as to a Procrustean bed, they compelled their cases to correspond. With the further progress of medicine came the fresh study of the old diseases and the discovery that much could be seen and demonstrated which it was difficult to reconcile with the old traditions. Then came an unsatisfactory period during which the old name was retained as expressing a real entity, and the attempt was made to divide and classify the varieties of this entity. Lastly comes the time when the old name is retained as a popular term, but it is recognized that it does not really designate a distinct form of disease. Such has been the history of such names as "cancer," "phthisis," and "Bright's disease."

I believe that the time has fully come to abandon the idea that there is such a disease as Bright's disease, and to cease from the attempt to describe varieties of a disease that does not exist. When we have divested our minds of this tradition we can begin to study the diseases of the kidneys and to try to classify them.

There seem to be three ways in which we can classify kidney diseases—according to their causes, according to the part of the kidney involved, or according to the nature of the morbid process.

The attempt to classify kidney diseases according to their causes is, in the present state of our knowledge, simply impossible.

The attempt to classify them according to the part of the kidney involved, to describe tubal nephritis, glomerulo-nephritis, interstitial nephritis, and arterio sclerosis, has been made many times. It has never given us a classification of any practical use for clinical purposes.

A classification according to the nature of the morbid process is altogether the most promising.

There are three morbid processes which occur in nearly every part of the body, which produce definite anatomical changes, cause regular clinical symptoms, and call for appropriate methods of treatment. These morbid processes are congestion, degeneration, and inflammation.

Congestion, whether acute or chronic, produces an accumulation of blood in the veins and capillaries of the part affected, causes local symptoms and disturbances of function, and is to be relieved by means addressed to the circulation of the blood.

Degeneration, whether acute or chronic, produces changes more or less profound in the parts affected; is regularly caused by poisons, by disturbances of circulation, and by other diseases; produces disturbances of function according to its severity; may be itself a cause of inflammation, and can be but little affected by any treatment.

Inflammation is attended with three essential features, which may occur separately or together—an escape of the elements of the blood from the vessels, a formation of new tissue, and a death of tissue. So we speak of exudative, productive, and necrotic inflammations.

Exudative inflammation is of short duration, leaves behind it no permanent changes in the parts affected, and can be favorably affected by treatment.

Productive inflammation runs an acute, subacute, or chronic course. It effects permanent changes in the inflamed parts. Its acute forms are very apt to become chronic. There is much variety as to the relative quantity of exudation and of new tissue. Treatment is not very satisfactory.

The different forms of kidney disease which are commonly included under the name of Bright's disease can all be conveniently classified under the heads of—

Congestion of the kidney.
Degeneration of the kidney.
Inflammation of the kidney.

Such a classification has the merit of being simple and easily understood, of resting on an anatomical basis, and of being of practical use for clinical purposes.

I. The Congestions of the Kidney.

These are naturally divided into acute and chronic congestions. They must always depend upon causes which affect the circulation and cause an accumulation of venous blood in the kidney.

Acute congestion of the kidney is produced by the ingestion of poisons, by extirpation of one kidney, by injuries, by surgical operations, and by unknown causes.

It may occur in kidneys previously normal, or in those already diseased.

The most marked symptom is the diminution or suppression of the urine, but albumin and casts may be present.

Such an acute congestion is a transitory condition, but when it occurs with injuries and with surgical operations it may apparently be a cause of death.

Chronic congestion of the kidneys is produced by some long-continued mechanical interference with the circulation of the blood, an interference which necessarily causes congestion of other parts of the body as well as of the kidneys.

The characteristic changes in the kidneys are: Swelling or flattening of the epithelium of the cortex tubes, dilatation and thickening of the capillaries of the glomeruli, congestion and dilatation of the pyramid veins.

The urine is more or less diminished in quantity according to the intensity of the congestion.

The specific gravity is from 1.020 to 1.025, but it may for a time fall to 1.010 or rise to 1.035. The excretion of urea is usually ten grains to the ounce; it may rise to twenty-one grains. Albumin and casts are absent, or only present in very small quantities. The bad results of chronic congestion are the diminished production of urine and the liability to the supervention of chronic nephritis.

II. Degeneration of the Kidneys.

Degeneration of the kidney is always a secondary process produced by the introduction into the body of inorganic poisons or of the poisons of the infectious diseases, or by the effect produced on the body by chronic diseases or vicious modes of life, or by disturbances of the circulation.

There are no changes in the kidneys except in the renal epithelium. In these cells are developed a variety of degenerative and necrotic changes.

It may happen that these degenerative changes are developed so rapidly that the bloodvessels are irritated, and there are added congestion and exudation of serum. We include, therefore, in this class both those kidneys the seat of degeneration alone, and those in which both degenerative and exudative inflammation exist, but the inflammation is secondary and subordinate to the degeneration.

Acute degeneration of the kidneys is found almost constantly with the infectious diseases and with poisoning by arsenic, phosphorus and mercury. It occurs in many different degrees of severity, and so we find the renal epithelium: merely swollen and granular, or infiltrated with granules, or broken and disintegrated, or in the condition of coagulation necrosis.

In correspondence with the degree of the degeneration of the renal epithelium is the severity of the clinical symptoms.

The urine is unchanged, or its quantity is diminished, or it contains a little albumin, or the albumin is abundant with casts and red and white blood-cells.

Many of the examples of this lesion are of so mild a type that they have no symptoms except the changes in the urine. The severe forms are dangerous to life; but dropsy and disturbances of the circulation are not associated with them.

Acute degeneration, therefore, includes a well-defined set of cases, definite in their lesions, their causes, their symptoms, and evidently not likely to be influenced by any treatment.

Chronic degeneration of the kidney is produced by the same mechanical causes as those which produce chronic congestion, by chronic alcoholism and by vicious modes of life. It is, therefore, always a secondary lesion.

The only changes in the kidney are in the epithelium of the tubes, of which the cells are swollen, granular, and infiltrated with fat. But if the degeneration is due to chronic congestion, there will also be changes in the glomeruli.

The quantity of the urine varies at different times in the same case and also in different cases; it may be abundant, scanty, or suppressed. Albumin and casts in moderate quantities are often present, but the specific gravity is not lowered.

Such a degeneration of the kidneys has a decided effect upon the health and nutrition of the patients. They lose flesh and strength, become anæmic, and finally pass into the typhoid state with delirium and stupor. Dropsy and disturbances of the circulation are not associated with this form of kidney disease. As the kidney lesion is always secondary to some other serious morbid condition, it is often difficult to tell how much of the loss of health is due to the primary disease and how much to the change in the kidneys.

III. The Inflammations of the Kidney.

These are naturally subdivided into—

A. Acute exudative nephritis.
B. Acute productive or diffuse nephritis.
C. Chronic productive or diffuse nephritis with exudation.
D. Chronic productive or diffuse nephritis without exudation.
Suppurative nephritis.
Tubercular nephritis.

A. *Acute Exudative Nephritis.*—This is an acute inflammation of the kidney, characterized by congestion, an exudation of plasma, an emigration of white blood-cells, and a diapedesis of red blood-cells from the vessels; to which may be added swelling or necrosis of the renal epithelium and changes in the glomeruli.

Of such a nephritis we may distinguish three varieties:

1. A mild form, which gives symptoms during life, but leaves no lesions in the kidney after death.

2. A more severe form, in which we find inflammatory changes in the kidney after death.

3. A form characterized by the excessive production of pus-cells.

Lesions. In a nephritis of this type we should expect that the inflammatory products, the serum, white and red blood-cells, and coagulable matter from the blood-plasma, would collect in the Malpighian bodies and tubes or infiltrate the stroma between the tubes; and that of the inflammatory products in the tubes and Malpighian bodies a part would be discharged with the urine and a part found in the kidney after death. We should also expect that the quantity of inflammatory products would be in proportion to the severity of the inflammation, and that an excessive number of pus-cells would belong to the especially severe forms of the disease. Still further it is evident that, with the milder examples of nephritis, with but little exudation, no inflammatory products might be found in the kidney after death, all having been discharged into the urine during life.

As a matter of fact, the kidneys do present just such changes. In the mild cases we find no decided lesions in the kidney after death. In the more severe cases the kidneys are increased in size, their surfaces are smooth, the cortical portion is thick and white, or white mottled with red, or the entire kidney is intensely congested. If the stroma is infiltrated with serum, the kidney is succulent and wet; if the number of pus-cells is very great, there will be little whitish foci in the cortex.

In such kidneys we find the evidences of exudative inflammation in the tubes, the stroma, and the glomeruli, all the changes being most marked in the cortical portion of the kidney.

The epithelium of the convoluted tubes is often simply flattened. As this same appearance is also found in the chronic congestion of heart disease, it seems probable that this change of the shape of the cells is merely due to the inflammatory congestion.

In other cases, not only is the epithelium flattened but there is also a real dilatation of the cortex-tubes. This dilatation does not involve groups of tubes, but all the cortex-tubes uniformly. In other cases, the epithelium of the convoluted tubes is swollen, opaque, degenerated, and detached from the tubes. But in fresh kidneys, properly preserved, this degeneration and desquamation of the epithelium is not nearly as constant or as marked a feature as would be supposed from the descriptions of some English observers. The tubes, whether with flattened epithelium or dilated, may be empty. More frequently, however, they contain coagulated matter in the form of irregular masses and of hyaline cylinders. The irregular masses are found principally in the convoluted tubes; they seem to be formed by a coagulation of substances contained in the exuded blood-plasma, and are not to be confounded with the hyaline globules so often found in normal convoluted tubes. The cylinders are more numerous in the straight tubes, but are also found in the convoluted tubes.

They also are evidently formed of matter coagulated from the exuded blood-plasma, and are identical with the casts found in the urine. The tubes may also contain red and white blood-cells.

In the cases in which there is an excessive emigration of white blood-cells, we find these cells in the tubes, in the stroma, or distending the capillary veins. This excessive emigration is not necessarily attended with exudation of the blood-serum, and so the urine of these patients may contain no albumin. The white blood-cells are not usually found equally diffused through the kidneys, but are collected in foci in the cortex. These foci may be very minute, or may attain a considerable size.

In the glomeruli we find changes which, at first sight, seem peculiar, but are really similar to the changes which we find in arteries and capillaries in many inflamed tissues. The cavities of the capsules may contain coagulated matter and white and red blood-cells, just as do the tubes. The capsular epithelium may be swollen, sometimes so much so as to resemble the tubular epithelium, and this change is most marked in the capsular epithelium near the entrance of the tubes.

The most noticeable change, however, is in the capillary tufts of the glomeruli. These capillaries are normally covered on their outer surfaces by flat, nucleated cells, so that the tuft is not made up of naked capillaries, but each separate capillary throughout its entire length is covered over with these cells. There are also flat cells which line the inner surfaces of the capillaries, although not uniformly, as is the case in capillaries in other parts of the body. Still, in spite of the presence of all these cells, the outlines of the walls of the capillaries are fairly distinct.

In exudative nephritis the swelling and growth of cells on and in the capillaries change the appearance of the glomeruli. They are larger more opaque; the outlines of the main divisions of the tuft are visible, but those of the individual capillaries are lost. It is difficult to tell how much these changes in the glomeruli interfere with the passage of the blood through their capillaries. In most cases of exudative nephritis the patients recover and the glomeruli return to their natural condition

In some examples of exudative nephritis we also find a thickening of the walls of the branches of the renal artery within the kidney. This thickening is principally due to a swelling of the muscle-cells in the walls of these vessels.

Etiology. Acute exudative nephritis is frequently a primary disease, either occurring after exposure to cold, or without discoverable cause. It complicates scarlatina, measles, diphtheria, typhoid fever, acute general tuberculosis, pneumonia, acute peritonitis, dysentery, erysipelas, diabetes, and many other of the infectious diseases and severe inflammations. It is one of the forms of nephritis which complicate the puerperal condition.

Symptoms. In the milder cases the only symptoms are the changes in the urine. This is somewhat diminished in quantity, of normal or high specific gravity; it contains albumin in moderate quantities, a few casts, sometimes blood.

In the more severe cases the changes in the urine are more decided. It is diminished in quantity, or even suppressed; its specific gravity is normal or high; the quantity of albumin is very large; the casts are numerous, hyaline, granular, containing white or red blood-cells or epithelium; there are also free white and red blood-cells and epithelial cells from the kidney and the bladder. As a rule, the quantity of albumin and the number of casts are in proportion to the severity of the nephritis, but this is not always the case. Large quantities of albumin, numerous casts, and many white and red blood-cells may be found in the urine of kidneys which, after death, show no structural changes; while, on the other hand, small quantities of albumin and a few hyaline casts are compatible with a severe nephritis. Still further, the number of casts found in the urine during life is not always in proportion to the number of casts and quantity of coagulated matter found in the corresponding kidneys after death.

In addition to the changes in the quantity and composition of the urine, the patients present constitutional symptoms which vary, in the different cases, as to their number and their severity. A febrile movement, with more or less prostration; stupor, headache, sleeplessness, restlessness, muscular twitchings, and general convulsions; dyspnœa, loss of appetite, nausea, and vomiting; a pulse of high tension with exaggerated heart-action, or hypertrophy of the left ventricle, dropsy and anæmia—these may be called the characteristic symptoms of acute exudative nephritis. Of these symptoms a certain number—the fever, the prostration, the loss of appetite and nausea, the anæmia, the diminution in the quantity of urine, the albumin and casts in the urine—are such as would naturally accompany an acute inflammation of the kidney, and very often they are the only symptoms which do accompany it.

But with this same kidney lesion a certain number of patients will present, besides the symptoms just mentioned, the additional features of cerebral symptoms, changes in the heart and circulation, and dropsy. We indeed might think that the cerebral symptoms and the dropsy were due to the diminished excretion of urine; but when these conditions occur, as they often do, in patients who are passing large quantities of urine of good specific gravity, and when they are absent, as they often are, in patients who are hardly passing any urine at all, it is evident that these symptoms are not directly due to the nephritis, but constitute a separate, complicating set of symptoms, which may be present or absent in any given case of the disease.

What the complicating lesion is which produces these symptoms we do not fully know; but the changes in the action of the heart and the dropsy naturally direct our attention to the arteries and capillaries, with the expectation of finding in them some morbid condition which will hinder the passage of the blood through them. Whether the morbid condition is of an inflammatory nature, or whether it is only a spasmodic contraction, we are as yet ignorant.

The cases of exudative nephritis, with an excessive production of pus-cells, have a somewhat different clinical history. Such a nephritis occurs both in children and adults; it may be primary, or complicate scarlatina, diphtheria, or measles.

The invasion is sudden, with a high temperature and marked prostration. Restlessness, delirium, headache, and stupor are soon developed and continue throughout the disease. The patients lose flesh and strength and pass into the typhoid state. Dropsy is slight or absent altogether. The urine is not so much diminished in quantity as one would expect; its specific gravity is not changed; albumin, casts, and red and white blood-cells are present in considerable quantities, but not always early in the disease, and may even be absent altogether.

Prognosis. It is a very fatal form of nephritis, and yet one easily misunderstood on account of the resemblance of its symptoms to those of an acute meningitis.

B. *Acute Productive or Diffuse Nephritis.*—This is the most serious and important of all the forms of acute nephritis, not only for the reason that it involves so many of the structures of the kidney, but because its lesions are from the first of a permanent character, and because disturbances of the circulation are so frequently associated with it. It is one of the forms of scarlatinal nephritis; it occurs early and late in the course of diphtheria; it is the most important variety of the nephritis of pregnancy, and it is especially frequent as a primary nephritis with or without a history of exposure to cold.

Lesions. The changes in the kidneys are extensive and well marked.

The kidneys are large, at first smooth, later sometimes a little roughened; the cortical portion is thick, white, or mottled with yellow or red, or congested; the pyramids are red.

In these kidneys we find the same lesions as have been described as belonging to exudative nephritis, but with two additional changes—changes which are found in the earliest stages of the inflammation and which give the characteristic stamp to the lesion; first, a growth of connective tissue in the stroma; second, a growth of the capsule-cells of the Malpighian bodies. Both these changes do not involve the whole kidney, but symmetrical strips or wedges in the cortex, which follow the line of the arteries. These wedges are small or large, few or numerous, regular or irregular, in the different kidneys. But in every wedge we find the same general characters: one or more arteries of which the walls are thickened; the Malpighian bodies belonging to this artery show an enormous growth of capsule-cells, with compression of the tufts; running parallel to these arteries a growth of connective-tissue cells and basement substance in the stroma. Between the wedges we find at first only the changes of exudative nephritis; later, a growth of diffuse connective tissue. Sometimes we find these wedges small, symmetrical, and at considerable distances from each other; more frequently they are much closer together, sometimes even becoming continuous.

If the nephritis is of acute type and recent, the new tissue between the tubes consists largely of cells; if the nephritis is of subacute type and longer duration, the tissue is denser and has more basement substance. Where the growth of new tissue is abundant, the tubes become small and atrophied.

In the wedges the constant change is the growth of new tissue in the stroma between the tubes, not an infiltration of the stroma with pus-cells, as in an exudative nephritis, but a new growth of cells and of basement substance. The tubes seem to become compressed and atrophied between the new tissue.

In each wedge is one or more of the arteries which run up into the cortex and give off the little branches ending in glomeruli. The walls of these arteries are thickened.

The glomeruli belonging to these arteries become the seat of changes of a permanent character. There is the same growth of the cells covering the vessels, and of the cells within them, as in exudative nephritis, reaching even a greater development. In addition, there is a growth of the cells lining the capsules to such a degree as to form a mass of cells compressing the tuft. The tuft apparently never returns again to its natural condition, but, as time goes on, the vessels are obliterated, the capsule-cells are changed into connective tissue, and the glomeruli are finally transformed into little balls of fibrous tissue.

Symptoms. The urine during the acute periods of the nephritis is scanty, colored by blood, of high specific gravity. It contains much albumin, numerous casts of all kinds, and red and white blood-cells. Exceptionally, the casts are few or absent. Later in the disease, or when it is of a more subacute type, the urine is more abundant, the specific gravity falls, the albumin and the casts continue. In the protracted cases the albumin and casts may for a time diminish or even disappear.

The patients develop all the symptoms which we are accustomed to associate with Bright's disease—headache, restlessness, neuralgic pains, delirium, stupor, coma, muscular twitchings and general convulsions, dyspnœa and cough, loss of appetite, nausea and vomiting, neuro-retinitis, diarrhœa, increased arterial tension and hypertrophy of the left ventricle of the heart, anæmia, loss of flesh and strength, and dropsy, often developed to an extreme degree.

The course of the disease varies with the intensity with which it is developed and the rapidity with which it proceeds.

The invasion may be sudden, the symptoms marked, and the duration of the disease short, with a fatal termination; or, with the same acute invasion, the disease may be protracted over many months; or the invasion may be gradual, the course of the disease subacute, the symptoms not continuous, but with periods of improvement or even apparent recovery, and the duration of the disease protracted for months and years.

The *prognosis* of this form of nephritis is bad. The lesion is a permanent and progressive one. It is, indeed, apparently possible to recover from it, although with damaged kidneys, but this is the exception. The rule is that, sooner or later, the disease proves fatal, although it may well happen that periods of improvement give rise to false hopes of recovery.

c. *Chronic Productive, or Diffuse Nephritis with Exudation.*—Although it is convenient to describe two forms of chronic nephritis—one with exudation and one without—yet it must be remembered that these are not separate lesions of the kidneys, but varieties of the same lesion. For, in all these kidneys, one form of inflammation—productive, with the formation of new tissue—is present. The exudation from the vessels is something which is added to this, but does not change it.

In speaking of the exudation of serum from the vessels and its presence in the urine, we speak of it as it occurs during the whole course of the disease, and not as it occurs for short periods.

We mean that in an exudative chronic nephritis there is usually a large quantity of albumin in the urine, but that in the protracted cases there may be periods during which the albumin diminishes or entirely disappears. In the same way in a non-exudative nephritis there may be periods during which albumin is present in considerable quantities. Generally speaking, the character of the clinical symptoms will vary with the presence or absence of the albumin.

Lesions. In chronic nephritis with exudation the size of the kidney is usually increased; there is a very extensive growth of connective tissue in the cortex; the renal epithelium is swollen, granular, degenerated, fatty, broken, or flattened; the tubes contain coagulated matter, cast matter or blood; the cortex tubes are atrophied in some places, dilated in others.

The glomeruli are changed in several different ways:

1. There is a growth of the capsule-cells in such numbers that they compress the tufts. The cells covering the capillaries are also increased in size and number. The capsule-cells may finally be changed into connective tissue and the tufts become atrophied.

2. The glomeruli are of large size, the cells covering the capillaries are increased in number so that the outlines of the capillaries are lost, but yet the capillaries are not compressed nor the glomeruli atrophied.

3. There is a growth of the cells which cover the capillaries and of the cells within them. Of the cells which cover the capillaries the cell-bodies become very large, the capillaries are compressed, and the glomeruli eventually become atrophied.

4. The walls of the capillary vessels become the seat of waxy degeneration, while the cells which cover them are increased in size and number.

5. If the nephritis follows chronic congestion, the capillaries are dilated and there is an increase in the size and number of the cells which cover the capillaries.

The arteries remain unchanged, or they are the seat of obliterating endarteritis, or there is a symmetrical thickening of all the coats of the artery, or all the coats of the artery are thickened and converted into a uniform mass of dense connective tissue, or there is waxy degeneration of the walls of the artery.

Causes. This form of nephritis occurs very frequently as a primary disease, especially in young adults. It follows acute diffuse nephritis, chronic congestion and chronic degeneration of the kidney. It may complicate syphilis, chronic phthisis, chronic endocarditis, prolonged suppuration, and chronic inflammation of the bones and joints.

Symptoms. The urine varies in quantity at different times. When the nephritis is most quiescent the quantity of urine is normal. During the exacerbations of the nephritis the urine is scanty or suppressed. When the patients are doing badly, often when they are dropsical, the quantity of urine is very much increased.

The specific gravity and the proportion of urea to the ounce of urine slowly diminish. In the cases of shorter duration the specific gravity is apt to run between 1.012 and 1.020. In the very chronic cases it will be between 1.001 and 1.005. Very low specific gravities regularly indicate a large growth of connective tissue in the stroma of the cortex, or waxy degeneration of the capillaries of the glomeruli.

The urine regularly contains albumin and casts. During the active periods of the disease the quantity of albumin is very large; during its quiescent periods it is smaller, and at times may entirely disappear. The number of casts varies in proportion to the quantity of albumin, but occasional exceptions to this rule are seen.

Dropsy may be considered a regular symptom of chronic exudative nephritis. It is rare to find a patient who goes through the disease without exhibiting this symptom. It may be developed at any time in the disease, continue uninterruptedly, or occur only in attacks. A peculiar pallor of the skin and white color of the sclerotic is seldom absent, and is quite characteristic of the disease. It corresponds to a diminution in the quantity of hæmoglobin and in the number of the red blood-cells. These changes in the blood are not, as a rule, far advanced; but sometimes they are, and some cases even die with the symptoms of pernicious anæmia.

Many of the patients are troubled with headache and sleeplessness. Acute uræmic attacks, with contraction of the arteries, convulsions, etc., may occur in the course of a chronic exudative nephritis, but they are of very much more frequent occurrence with the non-exudative form of the disease.

Chronic uræmia, on the contrary, is one of the ordinary ways in which an exudative nephritis proves fatal. The patients pass into a condition of alternating delirium and stupor, with a rapid, feeble, soft pulse.

Simple neuro-retinitis or nephritic retinitis are developed in a moderate number of cases.

Dyspnœa is a nearly constant symptom, but it is not always the same kind of dyspnœa, nor always produced by the same cause. It may be due to hydrothorax, to œdema of the lungs, to contraction of the arteries, or to failure of the heart's action.

In many patients the dyspnœa due to contraction of the arteries, or to failure of the heart's action, is the first symptom which attracts attention. It is a dyspnœa which comes on in attacks, especially at night and in the early morning, and is regularly worse when the patient lies down. It often begins while the patient is apparently in good health, but it is a sure premonition of serious disease.

A catarrhal bronchitis with cough and expectoration is often present.

Loss of appetite, nausea, and vomiting are frequent symptoms.

The heart is often affected. There may be hypertrophy of the left ventricle, dilatation of both ventricles, chronic endocarditis, myocarditis, or a feeble heart.

Course of the disease. There is hardly any limit to the variety of the disease, but the most constant symptoms are anæmia, dropsy, and albumin in the urine.

1. There are cases in which the symptoms are nearly continuous, the patients get steadily worse and die within one or two years. The anæmia, the dropsy, and the albumin are constantly present, and the patients die with dropsy or with chronic uræmia.

2. There are cases in which the anæmia, the dropsy, and the dyspnœa come on in attacks which last for weeks or months. Between the attacks the patients are comparatively well, often able to work, although the urine always contains albumin.

3. There are cases in which a number of years before death the patient has an attack of dropsy, etc., from which he apparently recovers and goes on able to work, but with urine of low specific gravity, which sometimes contains albumin. After an interval of many years comes the fatal attack with all the characteristic symptoms.

4. There are cases which for years have no symptoms but pallor of the skin and urine of low specific gravity containing albumin. These patients often for a long time feel so well that they cannot understand that they have a serious disease.

5. There are cases in which the first symptom is the attack of spasmodic dyspnœa, the patients otherwise feeling well. It may be months or years before the other symptoms are developed.

6. There may be a history of chronic endocarditis lasting for years before the renal symptoms are developed.

7. There are cases which apparently recover from the disease.

The *prognosis* is bad, but life may be prolonged for many years with but few symptoms, and recovery seems to be possible.

D. *Chronic Diffuse Nephritis without Exudation.—Lesions.* The larger number of these kidneys are found after death to be diminished in size; the two kidneys together may not weigh more than two ounces. The capsules are adherent, their surfaces are roughened or nodular, the cortex is thin and of red or gray color. A considerable number of the kidneys do not differ in their size or appearance from normal kidneys, except that the capsules are adherent and the surfaces of the kidneys roughened.

Many examples of chronic non-exudative nephritis follow chronic congestion. The kidneys then remain hard, but the cortex becomes thinned, the capsules adherent, and the surface roughened. Some of the kidneys, instead of becoming atrophied, are increased in size, weighing together from sixteen to thirty-two ounces. The surfaces of these large kidneys may be smooth or nodular; the cortex is thickened, its color is red, gray, or white. There is regularly a growth of new connective tissue in the cortex and also in the pyramids, which becomes more and more marked as the disease goes on. In the cortex the new tissue follows the distribution of the normal subcapsular areas of connective tissue, is in the form of irregular masses, or is distributed diffusely between the tubes. In the pyramids the growth of new connective tissue is diffuse.

The tubes, both in the cortex and pyramids, undergo marked changes. Those included in the masses of connective tissue are diminished in size; their epithelium is flattened, some contain cast matter, many are obliterated. The tubes between the masses of new connective tissue are more or less dilated; their epithelium is flattened, cuboidal, swollen, degenerated, or fatty. The dilatation of the tubes may reach such a point as to form cysts of some size, which contain fluid, or coagulated matter. These cysts follow the lines of groups of tubes, or are situated near the capsules.

Of the glomeruli a certain number remain of normal size, but with the tuft-cells swollen, or multiplied. Many others are found in all stages of atrophy until they are convered into little fibrous balls. The atrophy seems to depend partly on the growth of tuft-cells and intra-capillary cells, partly on the thickening of the capsules, partly on the occlusion of the arteries. If the chronic nephritis follows chronic congestion the glomeruli remain large, but with a marked growth of tuft-cells; or they become atrophied, but with the dilatation of the capillaries still evident. The capillaries of the glomeruli may be the seat of waxy degeneration.

The arteries exhibit the same changes as are found in chronic exudative nephritis.

Complicating lesions. Hypertrophy of the left ventricle of the heart is frequently caused by exudative nephritis, but still more frequently by the non-exudative form; but it must be admitted that such an hypertrophy, although frequent, is not constant, that both with exudative and non-exudative nephritis there may be no change in the wall of the left ventricle. The hypertrophy of the wall of the ventricle may after a time be succeeded by dilatation, or chronic degeneration, or myocarditis.

Chronic endocarditis is very frequently associated with chronic Bright's disease. The valvular lesions may cause chronic congestion, chronic degeneration, or chronic nephritis; or the same patient may suffer from chronic endocarditis, and either form of chronic nephritis, the lesions associated possibly due to the same causes, but not dependent on each other.

Pulmonary emphysema, chronic endocarditis, and cirrhosis of the liver, all of them examples of chronic productive inflammation, frequently accompany chronic nephritis.

With the endarteritis comes the additional danger of cerebral hæmorrhage.

Patients suffering with chronic nephritis are more liable than are other persons to attacks of pericarditis, bronchitis, and gastric catarrh.

Symptoms. The typical urine of chronic non-exudative nephritis is a urine increased in quantity, of a specific gravity of about 1.010, containing a diminished quantity of urea, without albumin or casts, or with a trace of albumin and a very few casts, except during exacerbations of the nephritis, when the quantity of albumin and the number of casts may be considerable. But very important modifications of the urine are of ordinary occurrence. It is quite possible, with nephritis of this type far advanced, to have urine not below 1.020 in specific gravity, and without albumin or casts. In such cases the diagnosis has to be made without reference to the urine. On the other hand, there are cases in which the specific gravity of the urine falls very low, almost to 1.000, either with or without waxy degeneration of the vessels. There are cases in which the quantity of urine is very much increased, several quarts in the twenty-four hours. During the attacks of contraction of the arteries, to which these patients are liable, the urine may be diminished to a few ounces or suppressed.

In a great many of the cases cerebral symptoms are developed at some time in the course of the disease. Headache and sleeplessness are often present, the headache sometimes so severe and continuous that the patient is nearly maniacal; or instead of the headache there are neuralgic pains in the different parts of the body.

Muscular twitchings and general convulsions are much more serious; they may be an early symptom, or not occur until late in the disease.

Hemiplegia, with or without aphasia, may be the first symptom to call attention to the nephritis, or may not occur until late in the disease. The invasion of the hemiplegia is sudden and is usually accompanied by coma. There is loss of motion alone, or of both motion and sensation.

The hemiplegia, coma, and aphasia may continue up to the time of the patient's death, or disappear after a few hours or days. In the latter case the patient may have several such attacks. These attacks have been ascribed to localized œdema of the brain. In the cases which I have seen there were no changes in the brain-tissue, but the cerebral arteries were damaged by chronic endarteritis.

Delirium, mild or violent, stupor, and coma may come on in sudden attacks, or be developed slowly and gradually.

When these cerebral symptoms come on in attacks, the arteries are contracted, the temperature is raised, and the patients are said to suffer from acute uræmia. Very often they recover from a number of these attacks. In the fatal attacks the pulse often loses its tension, but becomes rapid and feeble, and the patients die comatose, with a feeble heart.

Instead of such acute attacks of cerebral symptoms, the symptoms may come gradually in persons already much reduced by the kidney disease. The temperature is then apt to be below the normal, and the pulse is rapid and feeble.

Temporary blindness, neuro-retinitis, or nephritic retinitis, are developed in a moderate number of these patients.

Chronic bronchitis and emphysema very frequently exist, and their symptoms often form a large part of the clinical history.

The left ventricle of the heart is regularly hypertrophied. This by itself gives no symptoms; but if the arteries become contracted, or if the hypertrophied heart becomes feeble, then disturbances of the circulation are established which cause serious symptoms.

In the same way the complicating endocarditis which so often exists gives no trouble until the valves are a good deal changed, or the ventricles dilated, or the heart's action altered, or the arteries contracted; then the circulation is interfered with, and the results of venous congestion of different parts of the body show themselves.

Dyspnœa is a very frequent symptom, often the first symptom noticed by the patient. It is a spasmodic dyspnœa coming on in attacks which last for minutes, hours, or days. It is regularly made worse by mental or bodily exertion, or by the recumbent position. It does not resemble pulmonary asthma. It is apparently due to the association of changes in the arteries and in the heart. With contraction of the arteries alone, or with a feeble heart alone, no dyspnœa may exist; but if the contraction of the arteries is so great that the hypertrophied and laboring heart is unable to overcome the obstruction, or if with the contracted arteries the heart becomes dilated or feeble, then the attacks of dyspnœa begin. At first the attacks are not severe and are of short duration, but if the mechanical conditions which cause them cannot be controlled, they become longer and more distressing.

Perhaps the most striking examples of this dyspnœa are in the patients in whom it is the first symptom of the nephritis. They are apt to be middle-aged or elderly men, often engaged in large financial or commercial enterprises. They profess that they feel quite well and that they can attend to their affairs perfectly, but that they are very much annoyed because early every morning they have an attack of asthma. In spite of their professions of good health, it is evident that they are pale and that they have dyspnœa on exertion. The heart is found to be enlarged, with or without a murmur; its action is either labored or feeble. The pulse is tense. The urine is of a specific gravity of 1.010 to 1.030; it contains no albumin, or only a trace. In the earlier stages of the disease this dyspnœa can be controlled, but later on it is more distressing and difficult to remedy.

The stomach may become the seat of catarrhal gastritis or of spasmodic vomiting. But in some patients it continues to perform its functions fairly well.

Dropsy, as a rule, is absent with non-exudative nephritis, unless it is complicated by chronic endocarditis or cirrhosis of the liver.

Profuse bleeding from atrophied kidneys has been described by Bowlby in three cases.

Regularly, after a time, the nephritis exerts its effect upon the nutrition of the patient, and the flesh and strength are diminished. On the other hand, the patients do not become as pale as they do in exudative nephritis.

Course of the disease. It is characteristic of the chronic productive inflammations of the lungs, the heart, the arteries, the liver, and the kidneys, that, while they often exist as serious and fatal diseases, they also frequently exist as lesions which do not interfere with general good health and long life. This seems to depend, in part at least, on the rapidity with which the inflammatory changes in these different parts of the body are developed. If they are developed slowly enough the functions of the organ continue to be performed, in spite of the new growth of connective tissue in it.

So, with the kidneys, it is common enough to find chronic non-exudative nephritis far advanced in persons who die from accident or intercurrent disease, and have never given symptoms of renal disease.

In the same way we can often observe for years persons who have urine of low specific gravity, an hypertrophied left ventricle of the heart, and occasionally some increase of tension in the arteries, and who yet habitually enjoy very fair health.

But yet these same persons, if they are attacked with pneumonia or pericarditis, or suffer from a severe accident, will often develop serious or even fatal renal symptoms.

A very common form for the disease to take is that of attacks which are repeated a number of times, each attack worse than the preceding, and the general health more and more impaired between the attacks. During the attacks there are cerebral symptoms more or less severe—headache, sleeplessness, delirium, stupor, coma, convulsions. Dyspnœa may be present or absent. The arteries are contracted, with a tense pulse. There is loss of appetite, nausea, and vomiting. The urine is of low specific gravity and usually contains albumin. Between the attacks the patients at first seem to be fairly well, but later they gradually lose flesh and strength. The urine between the attacks is of low specific gravity and contains little or no albumin. They finally die in one of the attacks, feeble and emaciated.

In some of the patients spasmodic dyspnœa is the first symptom. This can often be controlled for months and years, and the patients then seem to be well. But after a time it is more difficult to manage, and other renal symptoms are added.

In some cases there are no symptoms for a long time, so that persons apparently in good health are attacked without warning by convulsions, coma, delirium, or hemiplegia. They may die in the first attacks or live to go through subsequent ones.

In some cases the only symptoms up to the time of the patient's death are gradual loss of flesh and strength and disturbance of digestion, the patient dying feeble and emaciated. These cases are hard to make out, unless the specific gravity of the urine is low and a little albumin present. Otherwise there is nothing to draw attention to the kidneys as the cause of the illness. Loss of eyesight from nephritic neuro-retinitis may be the first symptom.

The patients may suffer from the symptoms of cardiac disease for years before congestion or degeneration of the kidney is succeeded by chronic nephritis.

In many cases the course of the nephritis is modified by the complicating emphysema, phthisis, endocarditis, endarteritis, or cirrhosis of the liver.

TREATMENT.

Such a classification as that above given of kidney diseases brings with it a rational system of therapeutics.

Acute congestion of the kidneys can be relieved by the application of heat to the surface of the body.

Chronic congestion is best managed by the drugs which stimulate the heart and dilate the arteries.

We evidently have no means at our command by which we can influence acute degeneration of the renal epithelium; fortunately the great majority of the cases of acute degeneration are not serious.

Chronic degeneration also seems to be a condition which we are unable to treat.

In acute exudative and in acute diffuse nephritis the main indications for treatment are to diminish the severity of the nephritis and to regulate the circulation. To diminish the severity of the nephritis we employ cups over the lumbar region, heat over the lumbar region or over the entire body, and the internal use of calomel, sulphate of magnesia, opium, aconite, or digitalis.

The disturbances of the circulation are largely the causes of the cerebral symptoms and of the dropsy. With a laboring heart and contracted arteries we employ the drugs which dilate the arteries—chloral hydrate, opium, nitrate of amyl, and nitro-glycerin—or we diminish the quantity of the blood by venesection, sweating, or purging. With a feeble heart and relaxed arteries we use the cardiac stimulants.

In chronic nephritis climate and mode of life constitute the important parts of the treatment; it is doubtful if drugs exert any effect on the nephritis. A warm, dry climate and an out-of-door life are of the greatest importance. Medical treatment can, however, be employed with advantage for the relief of the anæmia, the dropsy, and the disturbances of circulation.

With the classifications of kidney disease now in common use you are all familiar. You know the number of names employed and the contradictory meanings attached to these names by different authors. Many of you must have experienced the extreme difficulty there has been in teaching students to understand Bright's disease. I leave it to you to determine how far the plan which I have proposed is likely to be of practical use.

CLINICAL OBSERVATIONS ON THE CARDIAC BRUITS OF CHLOROSIS.

By Alfred G. Barrs, M.D., M.R.C.P.,
SENIOR ASSISTANT PHYSICIAN TO THE GENERAL INFIRMARY AT LEEDS; LECTURER ON MATERIA MEDICA AND THERAPEUTICS IN THE YORKSHIRE COLLEGE OF THE VICTORIA UNIVERSITY.

In the medical out-patient department of the Leeds Infirmary, during the last two and a half years, I have been able to record the presence or absence of cardiac bruits in 205 cases of simple chlorosis. Cardiac bruits were present in 115 cases, and absent in 90 cases.

In the 115 cases in which bruits were present, their locality was recorded as follows:

A systolic bruit, audible at the base only,	in	56	cases
" " " apex "	"	13	"
" " " base and apex	"	24	"
" " " base, apex, and back,	"	22	"
		115	"